REMARKS

Claims 1, 4, 6, 19, 21, 31, 36, 38, 39 and 59-98 are pending. Claims 1, 4, 6, 19, 21, 31, 36, 38, 39 and 59-88 have been amended. New claims 89-98 have been added. Support for the amendments and new claims can be found throughout the application as originally filed. No new matter has been added.

Applicants also note that the Patent Office issued two forms regarding the power of attorney filed October 15, 2007. The first form is a Notice to Accept Power of Attorney. This is correct. The second form is a Notice Regarding Change of Power of Attorney. This form was erroneously sent to Fish & Richardson instead of the previous law firm. Therefore, Applicants ask that the Notice Regarding Change of Power of Attorney mailed October 22, 2007 be vacated and a new form sent to the previous law firm whose power was vacated.

Rejection Under 35 U.S.C. §103(a)

Claims 1, 4, 6, 19, 21, 31, 36, 38, 39 and 59-88 are rejected under 35 U.S.C. §103(a) as allegedly being unpatentable over Bolognesi et al. (1995), Barney et al. (2001) in view of Sivam et al. (1992) and Narazaki et al. (1996). The Office Action dismisses Applicants' previous arguments regarding Shugars et al. as not being "compelling considering the state of the art at the time of filing" and concludes that "the prior art clearly provides the requisite peptides of interest and teaches that conjugating said peptides through a maleimide intermediate increases the pharmacological profile of the peptide."

Applicants respectfully traverse this rejection. The rejection clearly ignores the state of the art regarding anti-fusogenic peptides and how one skilled in the art would expect an anti-fusogenic peptide to function in order to have anti viral effect. To clarify Applicants' arguments the claims have been amended to explicitly recite that the peptides being claimed are anti-fusogenic peptides. The importance of this property and the state of the art at the time of filing are discussed in more detail below.

For motivation to combine the cited references, the Office Action alleges that teachings of HSA attached to other proteins (such as lymphokines, erythropoietin, growth factors, etc. (as

disclosed by Sivam et al) or to a small organic molecule, bucillamine (as disclosed by Narazaki et al.), would motivate one skilled in the art to attach an anti-fusogenic peptide to HSA. But, based upon the knowledge in the art at the time of invention, one skilled in the art would not have been motivated to attach HSA to an anti-fusogenic peptide, much less expect that it would have anti-viral effect.

To understand how anti-fusogenic peptides such as those disclosed in Bolegnesi et al. and Barney et al. exhibit antiviral activity, the mechanism by which viruses such as HIV infect a target cell needs to be considered. Viral fusion proteins are typically spring loaded. At entry conditions, the spring snaps, and the viral protein changes shape (i.e. conformation). The formation of coiled-coil helical structures are key to this conformational change. Anti-fusogenic peptides are designed to interfere with the conformational change. However, for the anti-fusogenic peptide to work, it must fit into the coiled-coil or otherwise enter the structure of the viral proteins. Thus, the anti-fusogenic peptide has to fit into the coiled coil to exhibit antiviral effect.

At the time the above-referenced application was filed, one of ordinary skill in the art would have expected that these small peptides would not access their inhibitory sites if they are conjugated to a larger protein, such as serum albumin. Large proteins will collide with one another, causing steric hindrance and preventing coiled-coil formation by the inhibitory peptides.

In fact, at the time the above-referenced application was filed, the art actually taught that conjugates of such anti-fusogenic peptides did not maintain antiviral activity. Shugars et al, a paper from 1996 (prior to the filing of this case) (cited previously), teaches that an anti-fusogenic peptide conjugated to maltose binding peptide (MBP) did not maintain antiviral effect. Shugars et al. discuss the importance of the shape and size of anti-fusogenic peptides for antiviral activity. In order to study oligomerization of anti-fusogenic peptides, Shugars et al. fused an anti-fusogenic peptide (DP-107) to MBP. Shugars et al. report that while the anti-fusogenic peptide alone had antiviral activity, fusion proteins of the anti-fusogenic peptide and MBP did not have antiviral activity. Shugars et al. conclude that "***these results were not unexpected*** since the MBP carrier represents approximately 90% of the total protein mass and may sterically hinder the accessibility of the gp41 region for its target." (emphasis added). MBP is a protein

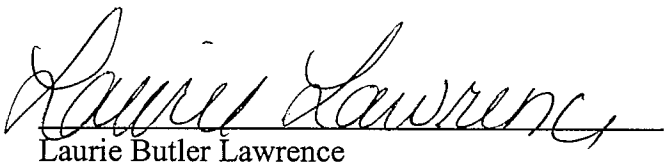
having a molecular weight of about 44 kDa. Albumin has a molecular weight of about 77 kDa, even larger than MBP.

For the reasons discussed above, a skilled artisan would not have been motivated to conjugate an anti-fusogenic peptide such as DP178 or an analog thereof the any protein, much less a protein the size of HSA, and expect such a conjugate to have anti-viral effect. Applicant were the first to understand and appreciate that the conjugates would function and have therapeutic antiviral effect. Therefore, Applicants respectfully request the withdrawal of this rejection.

The fees for the Petition for Extension of Time are being paid concurrently herewith on the Electronic Filing System (EFS) by way of Deposit Account authorization. Please apply any other charges or credits to Deposit Account No. 06-1050, referencing attorney docket no. 22102-002US1.

Respectfully submitted,

Date: 10/30/07



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